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Stable Ischemic Heart Disease

VULNERABLE CORONARY PLAQUES STILL EXIST EVEN IN LOW LDL-C LEVELS: EVIDENCE FROM STUDY WITH SERIAL CT CORONARY ANGIOGRAPHY

Poster Contributions
Hall C
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Session Title: Biomarkers, Predictors and Imaging in Stable Ischemic Heart Disease
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Background: Vulnerable plaques frequently develop coronary events in the presence of high low-density lipoprotein-cholesterol (LDL-C) levels. However, few data exist regarding the relationship between presence of vulnerable plaques and lipid profile in clinical settings. Therefore, we assessed the impact of LDL-C levels on appearance of the vulnerable plaques using CT Coronary Angiography (CTCA).

Methods and Results: We studied total 1056 patients (629 men, mean age 66±12 years) who underwent CTCA with 64-row cardiac multi-detector system for suspected coronary artery disease. When the vulnerable plaque defined as remodeling index (RI) >1.05 and CT value <50 HU was detected by CTCA, we administered statins such as rosuvastatin of 5-10mg/day or atorvastatin of 10-40 mg/day. Under these conditions, we re-evaluated the previously-determined coronary plaques and lipid profile 3 weeks later by CTCA. Definite vulnerable plaques were observed in 29 patients yielding LDL-C of 110±30 mg/dl, CT value of 21.7±16 HU, RI of 1.23±0.17. Among them, 14 patients treated with statins exhibited significant decreases in LDL-C from 121±31 to 65±24 mg/dl ($p<0.0001$), increases in CT value from 22.4±15.6 to 50.1±19.6 HU ($p=0.0003$) and decreases in RI from 1.22±0.12 to 1.09±0.07 ($p=0.003$). Importantly, in 3 patients among 4 patients with LDL- C less than 50 mg/dl, even after statin treatment, there still remained the vulnerable plaque exhibiting increased CT value from 12.0±1.3 to 38.0±11.0 HU ($p=0.055$), despite the marked reduction of LDL-C from 117±22 to 36±11 mg/dl ($p=0.010$).

Conclusions: These results demonstrate that some vulnerable plaques determined by CTCA still remain even after intensive lipid lowering therapy with statins, although a large part of plaques could be stabilized within 3 weeks after treatment. It should be further sought whether further manipulation targeting LDL-C <50 mg/dl is effective to stabilize the vulnerable plaques in clinical setting.